THE PATHOGENESIS OF REFLEXES, APROPOS OF A CASE OF TUBERCULOMA OF THE SPINAL MENINGES.

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The case which I report herewith presents some unusual features and suggests some inferences in regard to the pathogenesis of reflexes:

A. F., a colored girl, aged eighteen years, was admitted to the Philadelphia General Hospital, October 18, 1905, with the following history: About six months prior to her admission she noticed that her right arm was becoming weak and painful. Two weeks later the same condition developed in the left arm and shoulder. A month later, weakness without pain appeared in the lower extremities; this weakness gradually increased, so that within a month there was complete paralysis of the legs and of the right arm. At the same time there was loss of control of the bladder and rectum.

Upon examination there was found some preservation of power in the left arm, but the other three extremities were totally paralyzed. Wasting of the entire musculature of the body and especially of the extremities was quite pronounced. The paralysis of the legs was flaccid. The knee-jerks and Achilles-jerk were abolished; there was no ankle clonus, no Babinski sign. Test for sensations revealed that pain and touch sense were abolished in both legs as far up as the hips; the rest of the body presented normal sensations. (This statement is not absolute, as her answers varied from time to time.) There were multiple bed-sores. The pupils were unequal, the left being the larger; they reacted sluggishly to light and accommodation. Percussion and auscultation gave typical signs of tuberculous lungs. At no time did the patient complain of pain. She died two days after admission.

The postmortem record shows tuberculosis of the pleuræ and lungs, brown atrophy of the heart, and chronic interstitial nephritis. On the right side of the seventh cervical vertebræ was found a yellowish-gray mass. When the spinal canal was opened, a large cheesy-like mass was seen extending from the fifth cervical to the second dorsal vertebra on the right anterolateral surface of the dura. The dura with the growth was adherent to the bone. The vertebræ were softened; the greatest destruction was between the first dorsal and seventh cervical. On opening the dura the latter was found greatly thickened, its inner surface was smooth and glistening; the pia-arachnoid was smooth; the subarachnoid space contained a small amount of clouded yellowish fluid.

Microscopic examination shows that the greatest amount of damage done to the cord was at the lower cervical and upper dorsal region. (Fig. 1). On the side of the tumor is seen not only degeneration but also total destruction of the largest part of the white substance, particularly at the periphery, so that the nervous tissue is completely separated from the adjacent pia-arachnoid. There are a great many vacuoles and clefts in this portion of the white matter. The axis-cylinders in this area are either swollen or absent. The vacuoles are the result of complete destruction of the nerve fibers. On the opposite side the degeneration is confined to the entire periphery of the cord. Light, softened areas are also found in the posterior columns and in the white matter between and in front of the anterior cornua. The cornua themselves are deformed, very much diminished in size, with marked degenerative changes in their cells. Above this level, that is, in the upper cervical segments, there are distinct degenerated areas confined to the posterior colums, direct cerebellar, and partly Gowers' tracts (Fig. 2). This ascending degeneration extends upward as far as the first cervical segment, but no trace of pathological changes can be found in the medulla, in which the fillet and the restiform bodies are normal. The lower dorsal and lumbar segments of the cord show a descending degeneration in the crossed pyramid tract.

The most interesting changes are those of the meninges, particularly of the dura. Microscopically, the tumor was extradural and originated in the vertebræ. At the level of its maximum development the microscopic examination shows a very pronounced tuberculous pachymeningitis, invading not only the external aspect of the dura, but also penetrating in the intradural space, surrounding the root bundles of nerve fibers, and destroying them to a great extent. In some places portions of the tuberculous masses can be traced through the intradural space to the pia-arachnoid, where they produce an inflammation of the latter membrane, so that the pia presents in certain areas thickened portions filled with leukocytes. Close observation shows this infiltration continuing into the white substance of the cord and producing the above-mentioned disintegration. The leukocytic infiltration affects the bloodvessels of the entire section of the cord. The tuberculous neoplasm presents the usual characteristic features with the well-known lymphoid tissue and areas of necrosis.

The pathological process, in order of its development, therefore, can be summarized as follows: Tuberculosis of the vertebræ, formation of tuberculous tumors extraspinal and intraspinal, the latter on the external surface of the dura; tuberculous pachymeningitis; extension of the tuberculous process through the dura to the pia-arachnoid and through the latter into the cord itself.

The interesting points to which I wish to call special attention are the following:

1. Pain was only slight through the entire course of the disease

until admission to the hospital, when it had totally disappeared. The rule is that in meningeal tumors, pain is usually of great severity. The cause of the lack of pain in my patient was probably the absence of any marked involvement of the posterior roots. The latter were seen somewhat degenerated and covered with a slightly thickened pia only on the one side of the tumor.

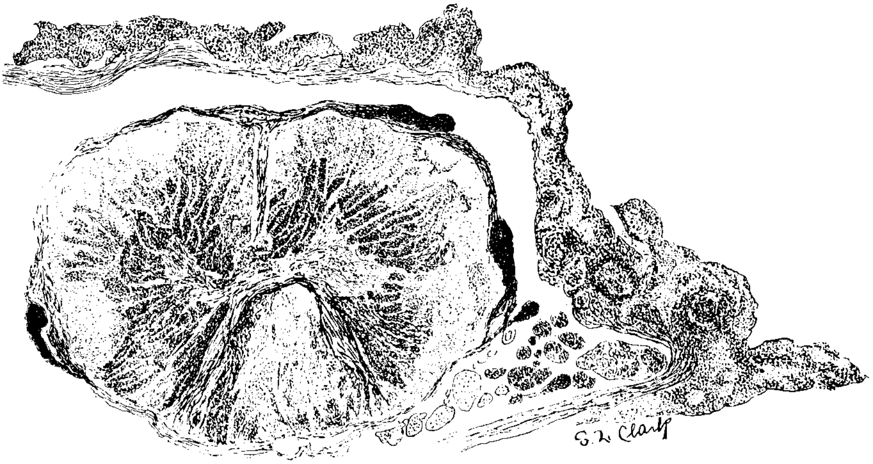

FIG. 1.—The level of greatest destruction of the spinal cord.

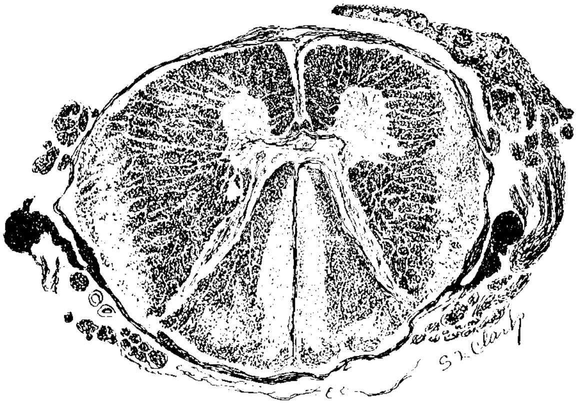

FIG. 2.—Above the level of greatest destruction of the spinal cord.

2. The tumor being extradural, places the case among the infrequent ones. Schlesinger, who made a statistical study of 400 tumors of the spinal cord, found only two extradural ones.

3. The tumor occupied the lower cervical portion, while the usual seat is the dorsal region. According to Collins' statistics of seventy cases, only fifteen were in the cervical region.

4. The extension of the pathological process could be traced through the dura and pia-arachnoid into the cord. The appearance of the sections at the level of greatest destruction of the cord tissue were those of tuberculous cords.[1] The vacuolation alongside of small masses of necrotic tissue in which is seen infiltration of round cells in the preserved small arterioles, and occasional giant cells, are all indications of tuberculous invasion of the cord itself. Although repeated attempts at staining for tubercle bacilli failed, this does not exclude the tuberculous condition of the cord tissue. Homen's[2] experiments prove conclusively that, while in recent cases the pathological changes of the nervous system depend upon the localization of the bacteria, in older cases in which the bacteria cannot be found, the alterations depend upon the poisons elaborated by the bacteria.

5. As a last interesting point, I wish to speak of the condition of the reflexes.

As is well known, a destruction of any segment of the cord may give flaccid or spastic paralysis. According to some neurologists the lumbar cord possesses an autonomous centre, so that the patellar reflex presupposes the integrity of the lumbar arc. If this arc is suddenly severed by a section of the cervical or thoracic segments, the knee-jerk disappears immediately after the shock; but later it will be reëstablished simultaneously with spasticity. The experiments of Ferrier, Sherrington, and Crocq are corroborative of this view.

Bruns, van Gehuchten, and Nonne advanced another theory. They say that in order to obtain a patellar reflex, not only the integrity of the lumbar cord, but also of certain cerebral centres is indispensable. When the lumbar centre is suddenly severed from its connection with the upper centres by a complete and rapid section of the cord, a flaccid permanent paralysis will ensue in spite of a descending degeneration of the pyramidal tract.

In chronic cases, that is, in cases with a slow and progressive destruction of a segment of the cord spastic paralysis appears at the onset and remains as such until the end. Raymond[3] calls attention to and reports two cases of a special tumor, psammoma, which is of a strictly localized nature, without tendency to generalization, and of a very slow evolution. This tumor originates in the meninges, compresses gradually a segment of the cord similarly to a solid, foreign body, and increases in size continually until complete destruction of the segment of the cord takes place. In such cases there is the best opportunity for observing a gradually oncoming and permanently remaining spasticity. Senator[4] also

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[1] See A. Gordon's case of Pott's disease, Jour. Nerv. and Ment. Dis., August, 1904.

[2] Neurol. Centralbl., 1904, No. 3, p. 21.

[3] Revue Neurol, 1902.

[4] Deut. Ztschr. f. klin. Med., 1897.

reports a case of psammoma of the cervical region, which produced a complete destruction of a cervical segment, and in which he observed spastic paralysis during the entire course of the affection.

In opposition to these facts I cite the facts of my case. The patient presented flaccidity during the entire course of her affection. Six months elapsed between the onset of the affection and death. This period is certainly more than is necessary for a descending lesion of the lateral columns to produce a spastic state of the paralysis, either by suppressing the inhibitory influence of the brain or by stimulating the cells of the anterior cornua. The destruction of the cord between the lower cervical and upper thoracic segments was, I may say, complete. The pathological process was slow and progressive.

That there is a centre in the lumbar cord, the above-mentioned experiments of Ferrier and others prove conclusively. But the question of an independent character of this centre presents certain objections. Assuming the existence of an autonomous centre in the lumbar cord, which in this case was totally severed from the encephalon, why did spasticity not develop? Is not this case an illustration of the dependency of the cord centre upon cerebral centres? I must conclude, therefore, that the reflex mechanism has multiple relations. The analysis of the accumulated observations tends more and more to show that the exclusivism of Charcot and Vulpian, on one hand, of Bastian, Crocq, Collier, and Buzzard, on the other, cannot be accepted, but that Grasset's views are more in conformity with facts—namely, that spinal, basilar, and cortical regions contain centres for reflexes and for tonicity.

SCLEROMA OF THE LARYNX.

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It is always of interest to record the phenomena of an unusual affection, and the study of scleroma of the larynx becomes the more interesting, for the reason that, like all diseases due to a bacillus, the contagious nature of the disease is to be considered, together with the possible need for recommending such action as will tend to prevent its spread.

We are all familiar with the interesting cases of scleroma of the nose, several of them having been recorded in our literature, but the case that I report herewith is the first instance of the affection beginning *de novo* in the larynx to be presented and published in North America. Like all cases seen in this country, this one had